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A delay differential equations model of plankton allelopathy

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Abstract

In this paper we have studied the dynamics of planktonic growth with special consideration on time dependent fluctuations in density of the species. We propose a modified delay differential equation model of the growth of two species of plankton having competitive and allelopathic effects on each other. The model system shows a stable limit cycle oscillation when the allelopathic effect is of a stimulatory nature.

Keywords: Plankton ecology; Allelopathy; Time delay; Hopf bifurcation; Limit cycle

1. Introduction

The study of tremendous fluctuations in abundance of many phytoplankton communities is an important subject in aquatic ecology. These changes of size and density of phytoplankton have been attributed to several factors, such as physical factors, variation of necessary nutrients, or a combination of these by various workers. Another important observation made by many workers is that the increased population of one species might affect the growth of another species or of several other species by the production of allelopathic toxins or stimulators, thus influencing seasonal succession [1].

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The toxin produced by the unicellular green alga, *Chlorella vulgaris*, is an autotoxin that not only limits the size of its own population [2,3] but also inhibits the growth of the planktonic algae *Asterionella formosa* and *Nitzschia frustulum* (Bacillareae) [4,5]. Pratt [6–8] studied a number of properties of a substance produced by *Chorella* and named it Chlorellin.

Besides the above work, evidence of widespread toxic inhibition of phytoplankton by other phytoplankton has been observed by many researchers [9–42]. Some of the genera of planktonic algae that were reported by the above researchers to have species inhibitory to other algae (in addition to those discussed above) were *Phormidium, Scenedesmus, Pediastrum, Cosmarium, Aphanizomenon, Micrasterias, Oscillatoria, Pandorina, Nostoc, Cylindrospermum, Mesotaenium, Aukistrodesmus, Anabaena, Microcystis, Ceratium, Asterionella, Haematococcus* (a motile green alga), *Chlamydomonas, Skeletonema, Olithodiscus, Peridinium, Gymnodinium, Ulva, Chorda, Ceramium, Ascophyllum, Chondrus, Fucus, Enteromorpha, Myriophyllum, Ceratophyllum, Lemna, Cladophora, Pithaphora, Hormotilla, Platydorina, Volvox, Eudorina, Gonium, Botrydium, Thalassiosira, Phaeodactylum, Scytonema* etc.

Some of these algae have also been observed to produce auxins which stimulate the growth of the other algae. For example, Berglund [24] has noted that the green alga, *Enteromorpha linza*, produces substances which are autostimulatory and stimulatory to the growth of *Enteromorpha* species. Monahan and Trainor [27] also found that the green alga *Hormotila blemista* stimulated its own growth and also stimulated one strain of *Scenedesmus*. Porter and Targitt [43] reported that Sponge (*Planktoris*) kills a coral through a waterborne allelochemic. Many other examples of toxic inhibition and auxic stimulation were observed in planktonic algae by several researchers. Such allelopathic stimulators and inhibitors certainly affect algal succession, blooms and pulses by causing stimulated (inhibited) species to have a selective advantage (disadvantage) in competition [1].

Most of the above work has been reported and discussed in detail in elegant reviews of Hellebust [42] and Rice [1]. Gupta and Houdeshell [44] dealt with the allelopathic effects of algae on other algae and allelochemic effects of algae on zooplankton in a differential-difference equations model of a dynamic ecosystem. Rice [1] has suggested that "all meaningful, functional ecological models will eventually have to include a category on allelopathic and other allelochemic effects".

Maynard Smith [45] incorporated the effect of toxic substances in a two species Lotka–Volterra competitive system by considering that each species produces a substance toxic to the other but only when the other is present. Chattopadhyay [46] investigated the stability properties of the above system, although the study contains the flaw of ignoring an important delay factor in the system. In reality, a species needs sometime for maturity to produce a substance which will be toxic (or stimulatory) to the other and hence a delay

term in the system arises. To our knowledge no such attempts have been taken to include the time delay effect in a model of allelopathic interaction in plankton ecology. The aquatic ecosystem might be more transparent if one can incorporate the time delay effect in the model system.

In this paper we have assumed that the production of allelopathic substance by the competiting species will not be instantaneous, but mediated by some time lag required for maturity of the species and from this viewpoint we have modified the model of Maynard Smith [45]. The biological meaning of the delay introduced into the model is that an allelochemical is produced by some diatoms in their mature phase of growth only. The delay thus accounts for the maturity time of the new born cells, after which they become capable of producing the allelopathic substance. We have investigated the local and global behaviour of this modified dynamical system. It has also been observed that time delay can drive the competitive system to sustained oscillations, as shown by Hopf bifurcation analysis and limit cycle stability, if the allelopathy is of a stimulatory nature. Hence the time delay effect produced by delayed toxin or auxin production can regulate the densities of different competing species in the aquatic ecosystem, thus influencing seasonal succession, blooms and pulses.

2. The mathematical model

Lotka–Volterra two species competition model can be written as

$$\begin{aligned}\frac{dN_1}{dt} &= N_1[K_1 - \alpha_1 N_1 - \beta_{12} N_2],\\ \frac{dN_2}{dt} &= N_2[K_2 - \alpha_2 N_2 - \beta_{21} N_1],\end{aligned} \tag{1}$$

where $N_1(t), N_2(t)$ are the population densities (number of cells per liter) of two competing species; K_1, K_2 the rates of cell proliferation per hour; α_1, α_2 the rate of intra specific competition of first and second species respectively; β_{12}, β_{21} the rate of inter specific competition of first and second species respectively and $K_i/\alpha_i (i = 1, 2)$ are environmental carrying capacities (representing number of cells per liter). The units of $\alpha_1, \alpha_2, \beta_{12}$ and β_{21} are per hour per cell and the unit of time is hours.

Maynard Smith [45] and finally Chattopadhyay [46] modified the system (1) by considering that each species produced a substance toxic to the other, but only when the other is present. Then the system (1) can be written as

$$\begin{aligned}\frac{dN_1}{dt} &= N_1[K_1 - \alpha_1 N_1 - \beta_{12} N_2 - \gamma_1 N_1 N_2],\\ \frac{dN_2}{dt} &= N_2[K_2 - \alpha_2 N_2 - \beta_{21} N_1 - \gamma_2 N_1 N_2],\end{aligned} \tag{2}$$

where γ_1 and γ_2 are, respectively, the rates of toxic inhibition of the first species by the second and vice versa.

Notice that the toxic effect of one species is dependent on the density of the other species so that the inhibiting effect is zero when either species is absent. In aquatic ecosystems it has been observed that some species of phytoplanktons also produce substances which stimulate the growth of other species. Hence we shall use the common terminology allelopathic substance or simply 'allelochemic' instead of toxic substance or toxin in our study. It seems also reasonable to assume that the production of the substance allelopathic to the competing species will not be instantaneous, but mediated by some discrete time lag required for maturity of the species. For simplicity, we introduce this time lag in the production of allelochemic by the first species only and then the system (2) will be reduced to

$$\begin{aligned}\frac{dN_1}{dt} &= N_1[K_1 - \alpha_1 N_1 - \beta_{12} N_2 - \gamma_1 N_1 N_2],\\ \frac{dN_2}{dt} &= N_2[K_2 - \alpha_2 N_2 - \beta_{21} N_1 - \gamma_2 N_1(t-\tau) N_2],\end{aligned} \tag{3}$$

where τ is the time (in hours) required for the maturity of the first species.

When $\gamma_1, \gamma_2 > 0$, the model system (3) represents an allelopathic inhibitory system, each species producing a substance toxic to the other; whereas when $\gamma_1 = -\bar{\gamma}_1, \gamma_2 = -\bar{\gamma}_2 (\bar{\gamma}_1, \bar{\gamma}_2 > 0)$, (3) represents an allelopathic stimulatory system, each species producing a substance stimulatory to the growth of the other species. We shall study both the systems in the following sections. We will assume throughout that solutions of (3) initiating in the first quadrant are positively invariant, continuable for all $t \geqslant 0$.

3. Equilibria and local stability

3.1. Case 1. Allelopathic inhibition (AI)

The steady state equilibria of the system (3) for $\tau = 0$ are as follows:

$$\begin{aligned} E_0&: \quad (0,0)\\ E_1&: \quad \left(\frac{K_1}{\alpha_1}, 0\right)\\ E_2&: \quad \left(0, \frac{K_2}{\alpha_2}\right)\\ E^*&: \quad (N_1^*, N_2^*). \end{aligned} \tag{4}$$

The non-zero equilibrium $E^*(N_1^*, N_1^*)$ can be determined by

$$p_{ij} N_i^2 + q_{ij} N_i + r_{ij} = 0, \quad i, j = 1, 2, \quad i \neq j, \tag{5}$$

where

$$
\begin{aligned}
p_{ij} &= \beta_{ji}\gamma_i - \alpha_i\gamma_j,\\
q_{ij} &= K_i\gamma_j - K_j\gamma_i - \alpha_i\alpha_j + \beta_{ij}\beta_{ji},\\
r_{ij} &= K_i\alpha_j - K_j\beta_{ij},\\
N_i^* &= \frac{1}{2p_{ij}}\Big(- q_{ij} \pm \sqrt{q_{ij}^2 - 4p_{ij}r_{ij}}\Big),
\end{aligned} \tag{6}
$$

with the conditions

$$
\begin{aligned}
&p_{ij} \neq 0,\\
&q_{ij}^2 - 4p_{ij}r_{ij} \geqslant 0,\\
&N_i^* > 0.
\end{aligned} \tag{7}
$$

For local stability analysis for all the equilibria of the system without delay see Chattopadhyay [46]. We shall study the local stability of (3). The perturbed system (3) around the equilibrium (N_1^*, N_2^*) can be written as

$$
\begin{aligned}
\frac{\mathrm{d}x}{\mathrm{d}t} &= Ax + By + a_{11}xy + a_{20}x^2 + a_{21}x^2y,\\
\frac{\mathrm{d}y}{\mathrm{d}t} &= Cx + Dy + Ex(t-\tau) + b_{11}xy + b_{02}y^2\\
&\quad + b'_{11}x(t-\tau)y + b'_{12}x(t-\tau)y^2,
\end{aligned} \tag{8}
$$

where

$$
\begin{aligned}
x &= N_1 - N_1^*,\\
y &= N_2 - N_2^*,\\
A &= K_1 - 2\alpha_1N_1^* - \beta_{12}N_2^* - 2\gamma_1N_1^*N_2^*,\\
B &= -N_1^*(\beta_{12} + \gamma_1N_1^*),\\
C &= -N_2^*\beta_{21},\\
D &= K_2 - 2\alpha_2N_2^* - \beta_{21}N_1^* - 2\gamma_2N_1^*N_2^*,\\
E &= -N_2^{*2}\gamma_2,\\
a_{11} &= -(\beta_{12} + 2\gamma_1N_1^*),\\
a_{20} &= -(\alpha_1 + \gamma_1N_2^*),\\
a_{21} &= -\gamma_1,\\
b_{11} &= -\beta_{21},\\
b_{02} &= -(\alpha_2 + \gamma_2N_1^*),\\
b'_{11} &= -2\gamma_2N_2^*,\\
b'_{12} &= -\gamma_2.
\end{aligned} \tag{9}
$$

Retaining only the linear terms in Eq. (8), the linearized system becomes

$$\frac{dx}{dt} = Ax + By,$$
$$\frac{dy}{dt} = Cx + Dy + Ex(t-\tau). \tag{10}$$

The system (10) is equivalent to

$$\frac{d^2x}{dt^2} - (A+D)\frac{dx}{dt} + (AD-BC)x - BEx(t-\tau) = 0. \tag{11}$$

We assume a solution of the form $x(t) = \beta e^{\lambda t}$ and $y(t) = e^{\lambda t}$. Therefore, $\beta = B/(\lambda - A)$ and we have the corresponding characteristic equation as

$$\Delta(\lambda, \tau) = \lambda^2 - (A+D)\lambda + (AD-BC) - BEe^{-\lambda\tau} = 0. \tag{12}$$

For the zero equilibrium $E_0(0,0), A = -K_1, B = 0, C = 0, D = K_2$ and $E = 0$. Therefore, the characteristic equation (12) becomes

$$\lambda^2 - (K_1 + K_2)\lambda + K_1K_2 = 0.$$

Hence $\lambda_{1,2} > 0$ and thus E_0 is always unstable.

For the axial equilibrium $E_1(K_1/\alpha_1, 0), A = -K_1, B = -(\beta_{12}K_1/\alpha_1 + \gamma_1K_1^2/\alpha_1^2), C = 0, D = K_2 - \beta_{21}K_1/\alpha_1$ and $E = 0$. Hence, the characteristic equation (12) becomes

$$\lambda^2 + \left(\frac{\beta_{21}K_1}{\alpha_1} + K_1 - K_2\right)\lambda + K_1\left(\frac{\beta_{21}K_1}{\alpha_1} - K_2\right) = 0,$$
$$(\lambda + K_1)\left(\lambda - K_2 + \frac{\beta_{21}K_1}{\alpha_1}\right) = 0,$$

Hence $\lambda_1 = -K_1$ and $\lambda_2 = K_2 - \beta_{21}K_1/\alpha_1$. Thus E_1 is

$$\text{locally stable if} \quad \frac{K_1}{K_2} > \frac{\alpha_1}{\beta_{21}},$$
$$\text{unstable (saddle) if} \quad \frac{K_1}{K_2} < \frac{\alpha_1}{\beta_{21}}. \tag{13}$$

In the similar way it can be shown that the other axial equilibrium $E_2(0, K_2/\alpha_2)$ is

$$\text{locally stable if} \quad \frac{K_2}{K_1} > \frac{\alpha_2}{\beta_{12}},$$
$$\text{unstable (saddle) if} \quad \frac{K_2}{K_1} < \frac{\alpha_2}{\beta_{12}}. \tag{14}$$

Hence combining (13) and (14), the condition required for the persistence of both the species is

$$\frac{K_i}{K_j} < \frac{\alpha_i}{\beta_{ji}} \quad (i, j = 1, 2;\ \ i \neq j). \tag{15}$$

It is to be noted that the time lag factor in (3) has no effect on the existence and stability properties of the zero and axial equilibria.

We now investigate the local and global stability of the interior (non- zero) equilibrium $E^*(N_1^*, N_2^*)$, $(N_1^*, N_2^* > 0)$, assuming (15), i.e., the zero and axial equilibria are unstable and thus repellent. For $N_1^*, N_2^* > 0$ we have

$$\begin{aligned} A &= -N_1^*(\alpha_1 + \gamma_1 N_2^*), \\ B &= -N_1^*(\beta_{12} + \gamma_1 N_1^*), \\ C &= -N_2^*\beta_{21}, \\ D &= -N_2^*(\alpha_2 + \gamma_2 N_1^*), \\ E &= -N_2^{*2}\gamma_2. \end{aligned} \tag{16}$$

Condition (15) gives $r_{ij} > 0$ in Eq. (3). Then system (6) has unique positive interior equilibrium if

$$\frac{\alpha_i}{\beta_{ji}} > \frac{\gamma_i}{\gamma_j} \quad (i, j = 1, 2;\ \ i \neq j)\ \ (\text{when } \gamma_1, \gamma_2 > 0). \tag{17a}$$

Combining Eqs. (15) and (17a) we have

$$\frac{\alpha_i}{\beta_{ji}} > \max\left(\frac{\gamma_i}{\gamma_j}, \frac{K_i}{K_j}\right) \quad (i, j = 1, 2;\ \ i \neq j)\ \ (\text{when } \gamma_1, \gamma_2 > 0) \tag{17b}$$

as the condition of existence of unique positive interior equilibrium of the system (3).

We now investigate the local and global attractive properties of the unique positive equilibrium $E^* = (N_1^*, N_2^*)$.

Substituting $\lambda = \alpha + \mathrm{i}\omega$ in Eq. (12) and separating real and imaginary parts, we obtain the system of transcendental equations

$$\begin{aligned} &\alpha^2 - \omega^2 - (A + D)\alpha + (AD - BC) - BE\mathrm{e}^{-\alpha\tau}\cos\omega\tau = 0, \\ &2\alpha\omega - (A + D)\omega + BE\mathrm{e}^{-\alpha\tau}\sin\omega\tau = 0. \end{aligned} \tag{18}$$

The stability or instability of the system is determined by the sign of those λ satisfying Eq. (12) if λ is real or the sign of α satisfying Eq. (18) if λ is complex.

To find the necessary and sufficient conditions for non-existence of delay induced instability we now use the following theorem [47].

Theorem 3.1. *A set of necessary and sufficient conditions for E^* to be asymptotically stable for $\tau \geqslant 0$ is the following:*

1. *The real parts of all the roots of $\Delta(\lambda, 0) = 0$ are negative.*
2. *For all real m and $\tau \geqslant 0$, $\Delta(\mathrm{i}m, \tau) \neq 0$, where $\mathrm{i} = \sqrt{-1}$.*

Theorem 3.2. *The unique interior equilibrium E^* of the system (3) with allelopathic inhibition is locally asymptotically stable for all $\tau \geqslant 0$.*

Proof. To prove this we shall use Theorem 3.1.

Step 1: From Eq. (12), we have

$$\Delta(\lambda,0)=\lambda^2-(A+D)\lambda+(AD-BC-BE)=0,$$
$$\lambda=\frac{1}{2}\left[(A+D)\pm\sqrt{(A+D)^2-4(AD-BC-BE)}\right].$$

Since, $(A+D)$ is always negative and

$$(A+D)^2-4(AD-BC-BE)=(A-D)^2+4(BC+BE)>0,$$

therefore $\Delta(\lambda,0)$ has no imaginary root. Moreover, we have the following cases:

(a) When $(AD-BC-BE)<0, \Delta(\lambda,0)=0$ has one positive and one negative roots.

(b) When $(AD-BC-BE)=0, \Delta(\lambda,0)=0$ has one zero and one negative roots.

(c) When $(AD-BC-BE)>0$, then both the roots of $\Delta(\lambda,0)=0$ are negative.

It can be readily verified from Eqs. (16), (17a) and (17b) that $(AD-BC-BE)>0$. Hence condition (1) of Theorem 3.1 is satisfied.

Step 2: Next we consider $\Delta(\mathrm{i}\omega_0,\tau_0)=0$ for real ω_0. Firstly, when $\omega_0=0$,

$$\Delta(0,\tau_0)=AD-BC-BE\neq 0.$$

Secondly, when $\omega_0\neq 0$, let

$$\begin{aligned}\Delta(\mathrm{i}\omega_0,\tau_0)=&-\omega_0^2-\mathrm{i}(A+D)\omega_0+(AD-BC)\\&-BE(\cos\omega_0\tau_0-\mathrm{i}\sin\omega_0\tau_0)=0.\end{aligned}$$

Separating real and imaginary parts we have the following:

$$\begin{aligned}&-\omega_0^2+(AD-BC)-BE\cos\ \omega_0\tau_0=0,\\&-(A+D)\omega_0+BE\ \sin\ \omega_0\tau_0=0.\end{aligned}\tag{19}$$

Squaring and adding both the equations of (19) we finally have

$$\omega_0^4+(A^2+D^2+2BC)\omega_0^2+(AD-BC+BE)(AD-BC-BE)=0.\tag{20}$$

As A^2+D^2+2BC is always positive, it is sufficient to consider the sign of $(AD-BC+BE)(AD-BC-BE)$ for existence of real roots of Eq. (20). Since $BE>0$ and $AD-BC-BE>0$ (see step 1), we must have $AD-BC+BE>0$. Hence, $\Delta(\mathrm{i}\omega_0,\tau_0)\neq 0$ for any real ω_0. This satisfies condition (2) of Theorem 3.1.

Therefore the unique interior equilibrium E^* of the allelopathic toxic (inhibitory) system is always asymptotically stable for all $\tau_0\geqslant 0$ and hence the delay is harmless in this case. □

3.2. Case 2. Allelopathic stimulation (AS)

In this case $\gamma_1 = -\bar{\gamma}_1$ and $\gamma_2 = -\bar{\gamma}_2(\bar{\gamma}_1, \bar{\gamma}_2 > 0)$, i.e., the last terms in the Eq. (3) give a positive effect instead of a negative effect as in case 1.

Here, except the interior (non-trivial) equilibrium, the local stability properties of all equilibria (axial and zero) and the criteria for persistence of both the species remain identical to case 1. In place of Eq. (5), the equation determining the interior equilibrium $\bar{E}^*(\bar{N}_1^*, \bar{N}_2^*)$ now becomes

$$p'_{ij}\bar{N}_i^{*2} + q'_{ij}\bar{N}_i^* + r'_{ij} = 0, \tag{21}$$

where $p'_{ij}, q'_{ij}, r'_{ij}$ are obtained from Eq. (6) by replacing γ_i by $-\bar{\gamma}_i(\bar{\gamma}_i > 0, i = 1, 2)$.

Since $r'_{ij} > 0$, the condition for the existence of a unique pair of positive equilibria $(\bar{N}_1^*, \bar{N}_2^* > 0)$, where both the species coexist, now becomes

$$p'_{12} < 0, \qquad p'_{21} > 0 \quad \text{and} \quad q'_{21} < 0 \tag{22a}$$

or

$$p'_{21} < 0, \qquad p'_{12} > 0 \quad \text{and} \quad q'_{12} < 0. \tag{22b}$$

From the values of p'_{ij} one can observe that the condition (22a) and obviously, not (22b), does not violate the condition of coexistence of both the species given by $\alpha_1\alpha_2 > \beta_{12}\beta_{21}$ (see condition (15)). Thus the condition (22a) is the only condition for the existence of unique pair of positive equilibria in the AS system given by Eq. (3) after replacing γ_i by $-\bar{\gamma}_i(i = 1, 2)$.

To investigate the local stability properties of $\bar{E}^*(\bar{N}_1^*, \bar{N}_2^*)$, we first note that the relations (16) now becomes

$$\begin{aligned} A &= -\bar{N}_1^*(\alpha_1 - \bar{\gamma}_1\bar{N}_2^*), \\ B &= -\bar{N}_1^*(\beta_{12} - \bar{\gamma}_1\bar{N}_1^*), \\ C &= -\bar{N}_2^*\beta_{21}, \\ D &= -\bar{N}_2^*(\alpha_2 - \bar{\gamma}_2\bar{N}_1^*), \\ E &= \bar{N}_2^{*2}\bar{\gamma}_2. \end{aligned} \tag{23}$$

Suppose $\lambda = \mathrm{i}\omega_0, \omega_0 > 0$, is a root of the characteristic equation (12) where A, B, C, D and E are given by Eq. (23). Now, we prove the following theorem.

Theorem 3.3. *If $A + D < 0$ and $B < 0$, then in the parametric region $BE < AD - BC < -BE$ the interior equilibrium $\bar{E}^*$ of the AS system is locally asymptotically stable for $0 < \tau < (A + D)/BE$ where ω_0 is defined in Eq. (26).*

Proof. From Eq. (12) it is clear that $\bar{E}^*$ is asymptotically stable if $AD - BC - BE > 0$ for $\tau = 0$. Hence it remains asymptotically stable for all $\tau > 0$. It can be observed that the eigenvalues of $\Delta(\lambda, \tau) = 0$ have negative real parts, provided one can guarantee that no eigenvalue with positive real part

bifurcates from infinity (which could happen since it is a retarded system). For stability analysis we require the Nyquist criterion and its consequences [48].

Consider the Eq. (11) and the space of all real valued continuous functions defined on $[-\tau,\infty)$ satisfying the initial conditions $x(t)=0$ for $-\tau\leqslant t<0, x(0^+)=P_1>0$ and $\dot{x}(0^+)=P_2>0$.

After taking the Laplace transformation of Eq. (11) and simplifying, we have

$$L(x(s))\equiv L(s)=\frac{P_1 s+P_2-(A+D)P_1}{s^2-(A+D)s+(AD-BC)-BE\mathrm{e}^{-\tau s}}. \tag{24}$$

The inverse Laplace transform of $L(s)$ will have terms which increase exponentially with t if $L(s)$ has any poles with positive real parts. Thus it is clear that a condition for stability of $\bar{E}^*$ is that all poles of $L(s)$ have negative real parts. We apply the Nyquist criterion [49] to see whether $L(s)$ has any poles to the right-half plane.

This criterion leads us to the conditions

$$\text{Im } \psi(\mathrm{i}\omega_0)>0, \tag{25}$$

$$\text{Re } \psi(\mathrm{i}\omega_0)=0, \tag{26}$$

where

$$\psi(s)=s^2-(A+D)s+(AD-BC)-BE\mathrm{e}^{-\tau s}, \tag{27}$$

with ω_0 the smallest positive value of ω for which Eq. (26) holds. Now,

$$\begin{aligned}\psi(\mathrm{i}\omega_0)=&-\omega_0^2-\mathrm{i}(A+D)\omega_0+(AD-BC)\\&-BE(\cos\omega_0\tau-\mathrm{i}\sin\omega_0\tau),\end{aligned} \tag{28}$$

$$\text{Im } \psi(\mathrm{i}\omega_0)=-(A+D)\omega_0+BE\sin\ \omega_0\tau \tag{29}$$

and

$$\text{Re } \psi(\mathrm{i}\omega_0)=-\omega_0^2+(AD-BC)-BE\cos\omega_0\tau=0. \tag{30}$$

Using Eqs. (25) and (26) on Eqs. (29) and (30), we have

$$\frac{A+D}{BE\tau}>\frac{\sin\omega_0\tau}{\omega_0\tau}$$

and

$$\omega_0^2=AD-BC-BE\cos\omega_0\tau.$$

Since $B<0$, we have, by restricting ω_0 to $0<\omega_0<\pi/\tau$,

$$AD-BC-BE>AD-BC-BE\cos\omega_0\tau>AD-BC+BE.$$

Hence $z=\omega_0^2$ and $z=AD-BC-BE\cos\omega_0\tau$ intersect on $0<\omega_0<\pi/\tau$. Since $(\sin\omega_0\tau)/\omega_0\tau<1$ on $0<\tau<\pi/\omega_0$ and since $A+D<0, BE<0$, the Nyquist criterion (25) is satisfied if $0<\tau<(A+D)/BE$.

From Eq. (30), we also have (in the parametric region $BE < AD - BC < -BE$)

$$AD - BC + BE < 0 < \omega_0^2 < AD - BC - BE \quad \text{for } 0 < \omega_0 < \frac{\pi}{\tau}, \tag{31}$$

so we have an upper bound ω_+ of ω_0, given by

$$\omega_+ = \sqrt{AD - BC - BE}. \tag{32}$$

Hence we can conclude that in our case the Nyquist criterion holds and the interior equilibrium $\bar{E}^*$ of the AS system is locally asymptotically stable for all values of τ satisfying $0 < \tau < (A + D)/BE$. □

4. Bifurcation of the solutions

In this section we state the condition under which the system goes through a point where the Hopf bifurcation occurs. We show the existence of such $\tau(= \tau_0)$ and $\omega(= \omega_0)$ where the Hopf bifurcation occurs.

Lemma 4.1. *If $A + D < 0$ and $BE < AD - BC < -BE$ there exists a unique pair of ω_0, τ_0, with $\omega_0, \tau_0 \geqslant 0, \omega_0\tau_0 < 2\pi$ such that $\Delta(\mathrm{i}\omega_0, \tau_0) = 0$, where ω_0, and τ_0 are given by Eqs. (33) and (38), respectively.*

Proof. From the analysis of the Theorem 3.2, we found from Eq. (20) that λ has a pair of purely imaginary roots of the form $\pm \mathrm{i}\omega_0$ provided $BE < AD - BC < -BE$.

The corresponding roots of Eq. (20) in this case are

$$\omega_0^2 = \tfrac{1}{2}[-(A^2 + D^2 + 2BC) + \{(A^2 + D^2 + 2BC)^2 - 4((AD - BC)^2 - (BE)^2)\}^{1/2}]. \tag{33}$$

Let suppose $\tau = \tau_0$ when $\omega = \omega_0$. From Eq. (19), we have

$$-\left[\frac{(BE)^2(1 - \cos^2\omega_0\tau_0)}{(A + D)^2}\right] + (AD - BC) - BE\cos\omega_0\tau_0 = 0$$

or,

$$(BE)^2\cos^2\omega_0\tau_0 - BE(A + D)^2\cos\omega_0\tau_0 + (AD - BC)(A + D)^2 - (BE)^2 = 0. \tag{34}$$

Let

$$f(z) = (BE)^2z^2 - BE(A + D)^2z + (AD - BC)(A + D)^2 - (BE)^2 = 0. \tag{35}$$

Therefore,

$$f(1) = (BE)^2 - BE(A+D)^2 + (AD-BC)(A+D)^2 - (BE)^2$$
$$= (A+D)^2(AD-BC-BE) > 0 \tag{36}$$

and

$$f(-1) = (BE)^2 + BE(A+D)^2 + (AD-BC)(A+D)^2 - (BE)^2$$
$$= (A+D)^2(AD-BC+BE) < 0. \tag{37}$$

Hence $f(z)$ has a real solution in $(-1,1)$ of the form $\cos\omega_0\tau_0 = k$, where $|k| < 1$.

From the second equation of (19)

$$\tau_0 = \frac{1}{\omega_0}\ \arcsin\ \frac{(A+D)\omega_0}{BE} + \frac{2n\pi}{\omega_0}, \quad n = 0,1,2,\ldots\ . \tag{38}$$

In Eq. (33) we assume $(AD-BC)^2 < (BE)^2$ [50], so that there is only one imaginary solution $\lambda = \mathrm{i}\omega_0(\omega_0 > 0)$ and therefore, the only crossing of imaginary axis is from left to right as τ increases and the stability of the trivial solution can only be lost and not regained. Obviously, in this case $n = 0$. □

Lemma 4.2. *For $\tau < \tau_0, \bar{E}^*$ is asymptotically stable. For $\tau > \tau_0, \bar{E}^*$ is unstable. Further as τ increases through $\tau_0, \bar{E}^*$ bifurcates into small amplitude periodic solutions.*

To prove this lemma we use the following lemma of G.J. Butler (see appendix 2 in Ref. [48]).

G.J. Butler's Lemma. *Let $A + D < 0, AD - BC > BE$. Then the real parts of the solutions of Eq. (12) are negative for $\tau < \tau_0$, where $\tau_0 > 0$ is the smallest value for which there is a solution to Eq. (12) with real part zero.*

Proof of Lemma 4.2. For $\tau = 0$, it is obvious that $\bar{E}^*$ is stable. Hence by G.J. Butler's Lemma, $\bar{E}^*$ remains stable for $\tau < \tau_0$. We have now to show that $\mathrm{d}\alpha/\mathrm{d}\tau|_{\tau=\tau_0} > 0$ when $\omega = \omega_0$, for $n = 0,1,2,\ldots$ This will signify that there exists at least one eigenvalue with positive real part for $\tau > \tau_0$, and hence $\bar{E}^*$ is unstable for $\tau > \tau_0$. Moreover, the condition for Hopf bifurcation [51] are then satisfied yielding the required periodic solutions. Now differentiating Eq. (18) with respect τ, we get

$$\{2\alpha - (A+D) + BE\tau\mathrm{e}^{-\alpha\tau}\cos\omega\tau\}\frac{\mathrm{d}\alpha}{\mathrm{d}\tau} + \{-2\omega + BE\tau\mathrm{e}^{-\alpha\tau}\sin\omega\tau\}\frac{\mathrm{d}\omega}{\mathrm{d}\tau}$$
$$= BE\mathrm{e}^{-\alpha\tau}\{-\alpha\cos\omega\tau - \omega\sin\omega\tau\} \tag{39}$$

and

$$\{2\omega - BE\tau\mathrm{e}^{-\alpha\tau}\sin\omega\tau\}\frac{\mathrm{d}\alpha}{\mathrm{d}\tau} + \{2\alpha - (A+D) + BE\tau\mathrm{e}^{-\alpha\tau}\cos\omega\tau\}\frac{\mathrm{d}\omega}{\mathrm{d}\tau}$$
$$= BE\mathrm{e}^{-\alpha\tau}\{\alpha\sin\omega\tau - \omega\cos\omega\tau\}. \tag{40}$$

Therefore,

$$[\{2\alpha-(A+D)+BE\tau e^{-\alpha\tau}\cos\omega\tau\}^2-(2\omega-BE\tau e^{-\alpha\tau}\sin\omega\tau)\{-2\omega+BE\tau e^{-\alpha\tau}\sin\omega\tau\}]\frac{d\alpha}{d\tau}=BE\tau e^{-\alpha\tau}[(-\alpha\cos\omega\tau-\omega\sin\omega\tau)\{2\alpha-(A+D)+BE\tau e^{-\alpha\tau}\cos\omega\tau\}-(\alpha\sin\omega\tau-\omega\cos\omega\tau)(-2\omega+BE\tau e^{-\alpha\tau}\sin\omega\tau)].$$

Now at $\alpha=0,\tau=\tau_0,\omega=\omega_0$, we have

$$\begin{aligned}&[\{-(A+D)+BE\tau_0\cos\omega_0\tau_0\}^2+(2\omega_0-BE\tau_0\sin\omega_0\tau_0)^2]\frac{d\alpha}{d\tau}\Big|_{\alpha=0,\tau=\tau_0,\omega=\omega_0}\\&=BE[-\omega_0\sin\omega_0\tau_0\{-(A+D)+BE\tau_0\cos\omega_0\tau_0\}\\&\quad+\omega_0\cos\omega_0\tau_0(-2\omega_0+BE\tau_0\sin\omega_0\tau_0)]\\&=BE\omega_0[(A+D)\sin\omega_0\tau_0-2\omega_0\cos\omega_0\tau_0]\\&=BE\omega_0\left[\frac{(A+D)^2\omega_0}{BE}-2\omega_0\frac{(-\omega_0^2+(AD-BC))}{BE}\right]\\&=\omega_0^2[(A+D)^2+2\omega_0^2-2(AD-BC)]\\&=\omega_0^2[2\omega_0^2+(A^2+D^2+2BC)]>0.\end{aligned}\tag{41}$$

Hence $d\alpha/d\tau|_{\alpha=0,\tau=\tau_0,\omega=\omega_0}>0$. Therefore, the transversality condition holds and hence Hopf bifurcation occurs at $\omega=\omega_0,\tau=\tau_0$. Hence the lemma. □

5. Stability of bifurcations

Here we determine a formula that establishes the stability of bifurcating periodic orbits. The calculation is based on Hassard et al. [52]. We assume the case where Hopf bifurcation occurs (at $\tau=\tau_0$ and $\omega=\omega_0$) and using the standard notation as in [52] we rewrite Eq. (8) in the form

$$\dot{x}_t=A_\alpha x_t+Rx_t,\tag{42}$$

where $x_t\in C([-\tau,0],\Re)$ is given by $x_t(\theta)=x(\theta-\tau)$; α represents the parameter values at $\tau=\tau_0,\omega=\omega_0$

$$A_\alpha\phi(\theta)=\begin{cases}\frac{d\phi}{d\theta} & -\tau\leqslant\theta<0,\\ \int_{-\tau}^{0}d\eta(\theta,\alpha)\phi(\theta) & \theta=0.\end{cases}\tag{43}$$

$$R\phi(\theta) = \begin{cases} \begin{pmatrix} 0 \\ 0 \end{pmatrix} & -\tau \leqslant \theta < 0, \\ \begin{pmatrix} a_{11}\phi_1(\theta)\phi_2(\theta) + a_{20}\phi_1^2(\theta) + a_{21}\phi_1^2(\theta)\phi_2(\theta) \\ \{b_{11}\phi_1(\theta)\phi_2(\theta) + b_{02}\phi_2^2(\theta) + b'_{11}\phi_1(\theta-\tau)\phi_2(\theta) \\ +b'_{12}\phi_2(\theta-\tau)\phi_2^2(\theta)\} \end{pmatrix} & \theta = 0, \end{cases} \tag{44}$$

$$\mathrm{d}\eta(\theta;\alpha) = \begin{pmatrix} A\delta(\theta) & B\delta(\theta) \\ C\delta(\theta) + \alpha\delta(\theta+\tau) & D\delta(\theta) \end{pmatrix} \mathrm{d}\theta. \tag{45}$$

An eigenfunction of the problem corresponding to the eigenvalue $\mathrm{i}\omega_0$

$$q(\theta) = \begin{pmatrix} \beta \\ 1 \end{pmatrix} \mathrm{e}^{\mathrm{i}\omega_0\theta}, \tag{46}$$

where

$$\beta = \frac{B}{\lambda - A}.$$

At $\lambda = \mathrm{i}\omega_0$

$$\beta = \frac{AB + \mathrm{i}B\omega_0}{A^2 + \omega_0^2}. \tag{47}$$

Now we define the standard inner product of ϕ and ψ as

$$\langle \psi, \phi \rangle = \overline{\psi(0)}\phi(0) - \int_{\theta=-\tau}^{0} \int_{\xi=0}^{\theta} \overline{\psi(\xi-\theta)}[\mathrm{d}\eta(\theta)]\phi(\xi)\mathrm{d}\xi. \tag{48}$$

So, to obtain the corresponding adjoint eigenfunction $q'(\theta)$ we use the standard result $\langle q', q\rangle = 1$ and $\langle q', \bar{q}\rangle = 0$, letting $q' = \mathrm{e}^{\mathrm{i}\omega_0\theta}(v_1, v_2)$, then we have

$$\begin{aligned} \langle q', q\rangle &= \overline{q'(0)}q(0) - \int_{-\tau}^{0}\int_{0}^{\theta} \overline{q'(\xi-\theta)}\mathrm{d}\eta(\theta)q(\xi)\mathrm{d}\xi \\ &= \beta\bar{v}_1 + \bar{v}_2 - \int_{-\tau}^{0}\int_{0}^{\theta} \mathrm{e}^{-\mathrm{i}\omega_0(\xi-\theta)}(\bar{v}_1 \ \bar{v}_2)\begin{pmatrix} 0 \\ \alpha\delta(\theta+\tau) \end{pmatrix}\begin{pmatrix} \beta \\ 1 \end{pmatrix}\mathrm{e}^{\mathrm{i}\omega_0\xi}\ \mathrm{d}\theta\ \mathrm{d}\xi \\ &= \beta\bar{v}_1 + \bar{v}_2 - \int_{-\tau}^{0}\int_{0}^{\theta} \mathrm{e}^{\mathrm{i}\omega_0\theta}\alpha\bar{v}_2\delta(\theta+\tau)\ \mathrm{d}\theta\ \mathrm{d}\xi \\ &= \beta\bar{v}_1 + \bar{v}_2 - \alpha\bar{v}_2(\tau\cos\omega_0\tau - \mathrm{i}\tau\sin\omega_0\tau). \end{aligned} \tag{49}$$

Therefore,

$$\beta\bar{v}_1 + \bar{v}_2(1 - \alpha\tau\cos\omega_0\tau + \mathrm{i}\alpha\tau\sin\omega_0\tau) = 1, \tag{50}$$

$$\langle q', \bar{q}\rangle = \bar{\beta}\bar{v}_1 + \bar{v}_2 - \int_{-\tau}^{0}\int_{0}^{\theta} e^{-i\omega_0(\xi-\theta)}(\bar{v}_1\bar{v}_2)\begin{pmatrix}0\\ \alpha\delta(\theta+\tau)\end{pmatrix}\begin{pmatrix}\bar{\beta}\\ 1\end{pmatrix}e^{-i\omega_0\xi}\,d\theta\,d\xi$$

$$= \bar{\beta}\bar{v}_1 + \bar{v}_2 - \int_{-\tau}^{0}\int_{0}^{\theta} e^{-i\omega_0(\xi-\theta)}\alpha\bar{v}_2\delta(\theta+\tau)e^{-i\omega_0\xi}\,d\theta\,d\xi. \tag{51}$$

Therefore,

$$\bar{\beta}\bar{v}_1 + \bar{v}_2 + i\alpha\frac{\bar{v}_2}{\omega_0}\sin\omega_0\tau = 0. \tag{52}$$

So, the required equations for $\bar{v}_1$ and $\bar{v}_2$ are

$$\beta\bar{v}_1 + e_1\bar{v}_2 = 1, \qquad \bar{\beta}\bar{v}_1 + e_2\bar{v}_2 = 0, \tag{53}$$

where

$$e_1 = 1 - \alpha\tau\cos\omega_0\tau + i\alpha\tau\sin\omega_0\tau,$$
$$e_2 = 1 + i\frac{\alpha}{\omega_0}\sin\omega_0\tau, \tag{54}$$

$$\bar{v}_1 = \frac{e_2}{e_2\beta - e_1\beta}, \qquad \bar{v}_2 = \frac{-\bar{\beta}}{e_2\beta - e_1\bar{\beta}}. \tag{55}$$

Finally, we have the values of v_1 and v_2 by taking the complex conjugate of Eq. (55). Using the notation as in Hassard et al. [52], we write

$$\begin{pmatrix}x\\ y\end{pmatrix} = zq + \bar{z}\bar{q} + W, \tag{56}$$

$$z = \left\langle q'\begin{pmatrix}x\\ y\end{pmatrix}\right\rangle, \tag{57}$$

$$\dot{z}(t) = i\omega_0 z(t) + \bar{q}'(0)\cdot f(w(z,\bar{z},\theta) + \mathrm{Re}\{z(t)q(\theta)\})$$
$$= i\omega_0 z(t) + \bar{q}'(0)\cdot f_0(z,\bar{z}), \tag{58}$$

where

$$f_0 = \begin{pmatrix}f_0^1\\ f_0^2\end{pmatrix}, \tag{59}$$

$$f_0^1 = \{W^1(0) + 2\ \mathrm{Re}(z(t)\beta)\}[a_{11}(W^2(0) + 2\mathrm{Re}\ z(t)) + a_{20}(W^1(0) + 2\ \mathrm{Re}(z(t)\beta)) + a_{21}(W^2(0) + 2\ \mathrm{Re}\ z(t))(W^1(0) + 2\ \mathrm{Re}\ (z(t)\beta))],$$

$$
\begin{aligned}
f_0^2 = \{&W^2(0) + 2\ \mathrm{Re}\ z(t)\}[b_{11}(W^1(0) \\
&+ 2\ \mathrm{Re}\ (z(t)\beta)) + b_{02}(W^2(0) + 2\ \mathrm{Re}\ z(t)) \\
&+ b'_{11}(W^1(-\tau) + 2\ \mathrm{Re}\ (z(t)\mathrm{e}^{-\mathrm{i}\omega_0\tau}\beta)) \\
&+ b'_{12}(W^1(-\tau) + 2\ \mathrm{Re}\ (z(t)\mathrm{e}^{-\mathrm{i}\omega_0\tau}\beta))(W^2(0) \\
&+ 2\ \mathrm{Re}\ z(t))].
\end{aligned} \tag{60}
$$

Using the result $\Delta(\mathrm{i}\omega, \tau) = 0$, for $\omega = \omega_0, \tau = \tau_0$, and letting $\Omega_0 = \mathrm{e}^{-\mathrm{i}\omega_0\tau_0}$ also we have assumed that $W = \mathrm{O}(|z|^2)$. We have retained only the terms necessary to compute $C_1(0)$. Therefore,

$$
\begin{aligned}
f_0^1 &= a_{11}(W^1(0) + \beta z + \bar{\beta}\bar{z})(W^2(0) + z + \bar{z}) + a_{20}(W^1(0) + \beta z + \bar{\beta}\bar{z})^2 \\
&\quad + a_{21}(W^1(0) + \beta z + \bar{\beta}\bar{z})^2(W^2(0) + z + \bar{z}) \\
&= (a_{11}\beta + a_{20}\beta^2)z^2 + (a_{11}\bar{\beta} + a_{20}\bar{\beta}^2)\bar{z}^2 + (a_{11}(\beta + \bar{\beta}) + 2a_{20}\beta\bar{\beta})z\bar{z} \\
&\quad + a_{21}\beta^2 z^3 + a_{21}\bar{\beta}^2\bar{z}^3 + 2a_{21}\beta\bar{\beta}(z^2\bar{z} + z\bar{z}^2) + \mathrm{O}(|z|^4),
\end{aligned} \tag{61a}
$$

$$
\begin{aligned}
f_0^2 &= b_{11}(W^1(0) + \beta z + \bar{\beta}\bar{z})(W^2(0) + z + \bar{z}) + b_{02}(W^2(0) + z + \bar{z})^2 \\
&\quad + (W^1(0) + (\beta z + \bar{\beta}\bar{z})\Omega_0)(b'_{11}(W^2(0) + z + \bar{z}) + b'_{12}(W^2(0) + z + \bar{z})^2) \\
&= (b_{11}\beta + b_{02} + b'_{11}\beta\Omega_0)z^2 + (b_{11}\bar{\beta} + b_{02} + \bar{\beta}b'_{11}\Omega_0)\bar{z}^2 \\
&\quad + (b_{11}(\beta + \bar{\beta}) + 2b_{02} + b'_{11}\Omega_0(\beta + \bar{\beta}))z\bar{z} \\
&\quad + b'_{12}\Omega_0(\beta z^3 + \bar{\beta}\bar{z}^3 + (2\beta + \bar{\beta})z^2\bar{z} + (2\bar{\beta} + \beta)z\bar{z}^2) + \mathrm{O}(|z|^4).
\end{aligned} \tag{61b}
$$

So, after taking the dot product of f_0 and $\bar{q}'(0)$ and after expanding, we have,

$$
\begin{aligned}
\dot{z} &= \mathrm{i}\omega_0 z + \bar{v}_1 f_0^1 + \bar{v}_2 f_0^2 \\
&= \mathrm{i}\omega_0 z + \tfrac{1}{2}g_{20}z^2 + \tfrac{1}{2}g_{02}\bar{z}^2 + g_{11}z\bar{z} + \tfrac{1}{6}g_{30}z^3 + \tfrac{1}{6}g_{03}\bar{z}^3 + \tfrac{1}{2}g_{21}z^2\bar{z} \\
&\quad + \tfrac{1}{2}g_{12}z\bar{z}^2 + \mathrm{O}(|z|^4),
\end{aligned} \tag{62}
$$

where

$$
\begin{aligned}
g_{20} &= 2[\bar{v}_1(a_{11}\beta + a_{20}\beta^2) + \bar{v}_2(b_{11}\beta + b_{02} + b'_{11}\beta\Omega_0)], \\
g_{02} &= 2[\bar{v}_1(a_{11}\bar{\beta} + a_{20}\bar{\beta}^2) + \bar{v}_2(b_{11}\bar{\beta} + b_{02} + b'_{11}\bar{\beta}\Omega_0)], \\
g_{11} &= \bar{v}_1(a_{11}(\beta + \bar{\beta}) + 2a_{20}\beta\bar{\beta}) + \bar{v}_2(b_{11}(\beta + \bar{\beta}) + 2b_{02} + b'_{11}(\beta + \bar{\beta})\Omega_0), \\
g_{12} &= 2[2\bar{v}_1 a_{21}\beta\bar{\beta} + \bar{v}_2 b'_{12}\Omega_0(\beta + 2\bar{\beta})], \\
g_{21} &= 2[2\bar{v}_1 a_{21}\beta\bar{\beta} + \bar{v}_2 b'_{12}\Omega_0(2\beta + \bar{\beta})], \\
g_{30} &= 6(\bar{v}_1 a_{21}\beta^2 + \bar{v}_2 b'_{12}\beta\Omega_0), \\
g_{03} &= 6(\bar{v}_1 a_{21}\bar{\beta}^2 + \bar{v}_2 b'_{12}\bar{\beta}\Omega_0).
\end{aligned} \tag{63}
$$

Finally, we use the expression of Hassard et al. [52]

$$C_1(0) = \frac{\mathrm{i}}{2\omega_0}\left(g_{20}g_{11} - 2|g_{11}|^2 + \frac{1}{3}|g_{02}|^2\right) + \frac{1}{2}g_{21},$$

$$\mu_2 = -\frac{\mathrm{Re}\, C_1(0)}{\dot{\alpha}(0)}. \tag{64}$$

So, the bifurcation is supercritical if $\mu_2 > 0$ and subcritical if $\mu_2 < 0$.

6. Numerical results

In this section we give some numerical results based on the formulae in Sections 3–5. These are obtained for the values $K_1 = 2, K_2 = 1, \alpha_1 = 0.07$, $\alpha_2 = 0.08, \beta_{12} = 0.05, \beta_{21} = 0.015, \gamma_1 = -\bar{\gamma}_1 = 0.0008, \gamma_2 = -\bar{\gamma}_2 = 0.003$. (For a discussion on parameter values and their appropriateness in plankton allelopathy, see Section 7.) Regarding the stability calculations in Section 5, we note that Eqs. (47), (63) and (64) yield (for the chosen set of parameter values) the values $\beta = 0.7868 - 0.4821\mathrm{i}$, $v_1 = 0.00218 + 0.00396\mathrm{i}, v_2 = -0.00173 - 0.00108\mathrm{i}, g_{20} = -0.000281$, $g_{02} = 0.000066 - 0.000234\mathrm{i}$, $g_{11} = -0.000234 - 0.000188\mathrm{i}, g_{21} = -0.0000008 - 0.00000065\mathrm{i}, \mathrm{Re}\, C_1(0) = -0.000000425$.

Since $\dot{\alpha}(0) > 0$ (from Eq. (41)), we have $\mu_2 > 0$. Thus the bifurcation is supercritical and the system exhibits a stable limit cycle.

Numerical solutions of Eq. (3) with both $\gamma_i > 0$ and $\gamma_i < 0$, i.e., both of the AI and AS systems are also presented (Figs. 1–3) taking the unit of t and τ in hours.

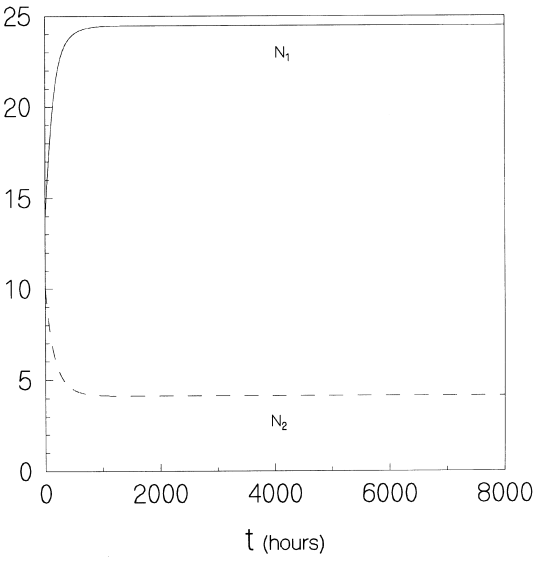

Fig. 1. Solution of the Al system for all $\tau \geqslant 0$.

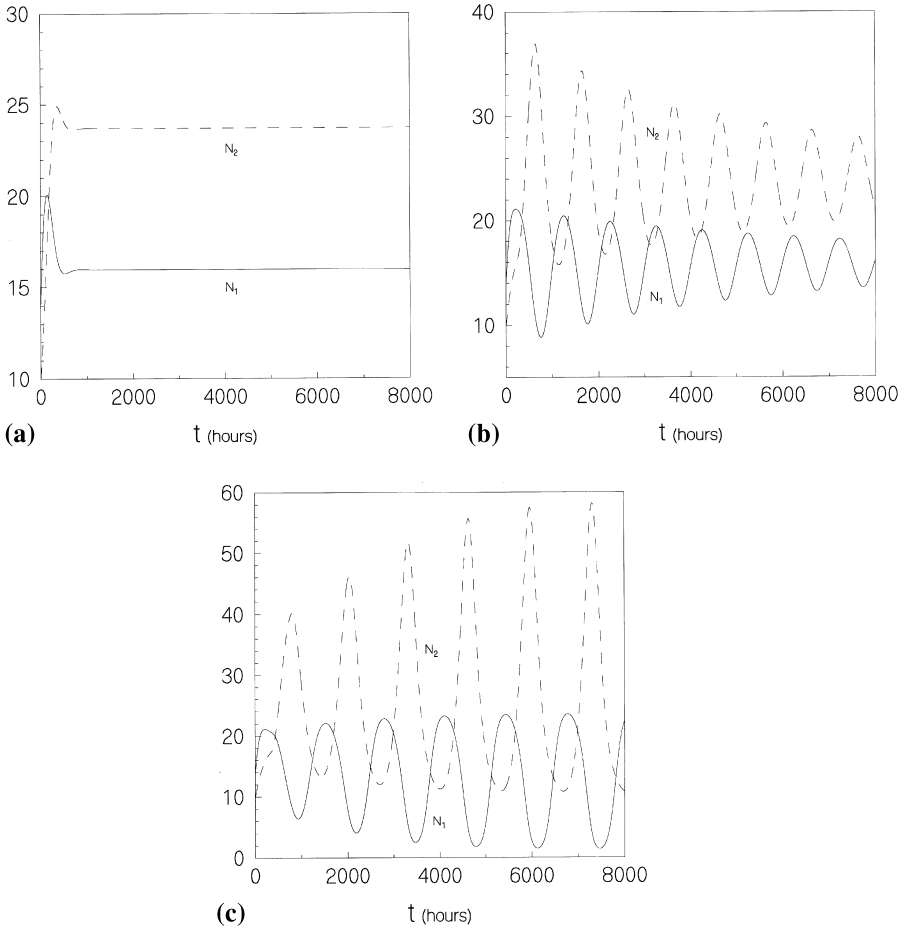

Fig. 2. (a) Solution of the AS system in absence of delay ($\tau = 0$). (b) Asymptotically stable solution (with decaying oscillation) of the delayed AS system for $\tau < \tau_0 = 290$ h. (c) Unstable solution (with growing oscillation) of the delayed AS system for $\tau > \tau_0 = 290$ h.

Numerical integration was carried out using modified fourth-order Runge–Kutta method. The results indicate that the equilibrium solution is stable for all $\tau \geqslant 0$, for the chosen parameter values, when the AI system ($\gamma_i > 0$) is considered (Fig. 1). With the same set of parameter values (expecting only replacing γ_i by $\bar{\gamma}_i$ in Eq. (3)) the equilibrium solutions of the AS system indicate stability (by decaying oscillation) for $0 \leqslant \tau < 290$ and instability (by growing oscillation) at $\tau > 290$ (Fig. 2(a)–(c)). The AS system exhibits a limit cycle stable periodic solution at the bifurcation value $\tau_0 = 290$ h $= 12.1$ days (approx.). The large amplitude stable periodic solutions and limit cycle trajectory in the

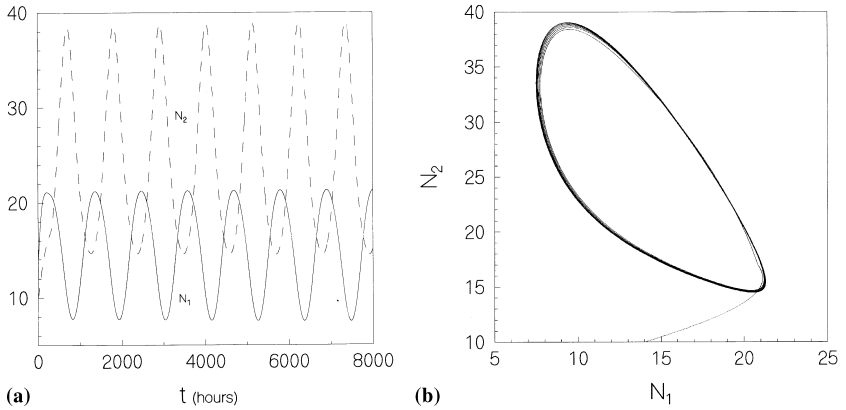

Fig. 3. (a) Bifurcating stable limit cycle solution of the delayed AS system $\tau = 290$ h (i.e., approx. 12.1 days). (b) Phase portrait of stable limit cycle trajectory of the delayed AS system with $\tau = 290$ h (i.e., approx. 12.1 days).

$N_1 - N_2$ plane are demonstrated in Fig. 3(a) and (b). Fig. 3(a) shows a typical model-genarated stable pattern of blooms over a one year span. Eight successive blooms of each species occurring alternatively with each other are exhibited annually. Since this is a stable limit cycle, this pattern repeats each year.

7. Discussion

In this paper we have attempted to study algal allelopathy with the help of a modified Lotka–Volterra type competition of two coexisting phytoplankton communities. Allelopathic effect in the model has been incorporated by introducing the production of an allelopathic substance by each of the species. The new-born plankton cells take some maturity time to produce effective allelochemicals, thus inducing a time-lag effect in the model.

It is observed that delay of all dimensions does not induce any instability, or stated otherwise, the delay is totally harmless if the allelopathy is of an inhibitory (toxin) type. On the other hand, delay of certain dimension destabilizes the system and leads to limit cycle stable periodic solutions through Hopf Bifurcation, if the model system is of a stimulatory (auxin) type.

The stability of the limit cycle periodic solution has been analytically proven, by using the method given in Hassard et al. [52]. Numerical solution and computer simulation of the model system have also been obtained to substantiate the analytical results.

The postulate that allelopathic interaction, both toxic and auxic, maintains a balanced phytoplankton ecology, has been evidenced by many experimental findings. Allelopathy has been described as one of the factors that controls

blooms, pulses and succession in the abundance of phytoplankton species in all kinds of bodies of water. Although extensive experimental study on allelopathic effects in planktonic world has been carried out, studies based on mathematical models on this important subject are absent or rare in the literature.

For the numerical analysis the values of the growth terms K_1 and K_2 are set to 2 and 1, respectively. This means that the growth term is reasonable 2 and 1 cell division per hour for the carrying capacity $K_1/\alpha_1 \approx 30$ and $K_2/\alpha_2 = 12.5$, respectively (these values of K_1/α_1 and K_2/α_2 represent over approximately 14 000 and 6000 cells per liter which are the theoretical maximum densities of the two species, respectively). Other parameters such as crowding (intra-species competition) coefficient, inter-species competition coefficient, inhibiting (or stimulating) allelopathic coefficients denoted by $\alpha_i, \beta_{ij}, \gamma_i(-\gamma_i)(i, j = 1, 2, i \neq j)$, respectively, have been chosen appropriate to plankton allelopathy. Actual values of these parameters can be estimated from experimental observations and collection of data set of cell counts over a long period. The magnitude of the time delay at which the system bifurcates into a stable limit cycle solution is of the order of 290 h, i.e., 12.1 days approximately for the chosen set of parameters. The present numerical analysis is based on purely hypothetical set of parameters. The aim of this study was to investigate the delayed allelopathic effect on the bloom, seasonal succession and pulses in aquatic ecosystem with the help of a mathematical delay model. If the growth rates K_i's are allowed to vary over a twelve month cycle, i.e., by substituting K_i by $K_i(1 - K_i' \cos(t/6))$, where K_i' regulate the amplitude of the annual oscillation and t measures time in months, the limit cycle is periodically coupled to an external forcing that represents the effect of an annual change in temperature on cell division rate. This coupling gives rise to an irregular pattern of bloom severity from year to year [53] as is actually observed in nature. Although the model results may not yield a high correlation with the actual field data because of the presence of other environmental noise, a modest correlation will suggest that even if the model does not replicate actual bloom events it lends support to the role of allelopathic effects in regulating bloom dynamics.

The present theoretical study shows that if the allelopathic model investigated by us is of a stimulatory type (AS system), it can successfully exhibit periodic fluctuations in plankton populations. On the other hand if the model investigated is of an inhibitory type (AI system), periodic fluctuation cannot be exhibited by it.

The failure of demonstrating fluctuation in the AI system, may be, because of the reason that the toxic allelopathic mechanism existing in the real world has not been properly captured in the present model. After a long series of experiments on fresh water phytoplankton allelopathy, Akehurst [54] suggested that phytoplankton might produce substances that suppress the growth of some species of phytoplankton and activate the growth of the others. It may

be that some species of phytoplankton, while being toxic to the others, may also be autotoxic themselves. Or, some species may produce substances which are toxic or stimulatory to the others while they themselves do not experience any reciprocal effects. Hence all the actual allelopathic mechanisms existing in the phytoplanktonic world may not be captured in a single mathematical model. An attempt to do this, although it may be a formidable task, is nevertheless, worth pursuing. However, the simple model presented here can adequately demonstrate the dynamics of a competitive and allelopathically interacting two species planktonic ecosystem.

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